

CENTERS FOR DISEASE CONTROL

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CDC ***Surveillance*** ***Summaries***

MORBIDITY AND MORTALITY WEEKLY REPORT

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U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
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Foreword

The purpose of the *CDC Surveillance Summaries* is to make available the most current information on conditions of public health interest for which CDC has major responsibility. The *CDC Surveillance Summaries* are published quarterly and provide detailed analysis of the most current available data obtained for CDC surveillance programs. These reports complement other data published by CDC in the *Morbidity and Mortality Weekly Report (MMWR)*, the *MMWR Annual Summary*, and various disease-surveillance reports. This volume contains epidemiologic information derived from surveillance forms, special investigations, and other sources of information collected at the state and national levels.

History of CDC Surveillance Activities

CDC has been actively involved in disease-surveillance activities since the formation of the Communicable Disease Center in 1946. The original scope of the National Surveillance Program included the study of malaria, murine typhus, smallpox, psittacosis, diphtheria, leprosy, and sylvatic plague. In 1954, a surveillance section was established within the Epidemiology Branch of CDC, primarily concerned with planning and conducting continuing surveillance and making periodic reports. National emergencies such as the Asian influenza pandemic and the discovery of Legionnaires' disease have prompted the involvement of CDC in new surveillance activities. Over the years the surveillance activities of CDC have expanded to include not only new areas in infectious disease but also programs in human reproduction, environmental health, chronic disease, risk reduction, and occupational safety and health. Ongoing evaluation of these programs has led to new methods of data collection and analysis and has prompted examination of how data are disseminated to the public health community.

In 1980 and 1981, a survey of CDC staff and state epidemiologists suggested that improved coordination of surveillance reports with the *MMWR* and the *MMWR Annual Summary* would facilitate timely publication; provide greater uniformity in the acquisition, evaluation, and reporting of surveillance data; and encourage use of these data. Several approaches to the development of a systematic process of disseminating disease-specific surveillance reports were considered. On the basis of considerations of timeliness, cost advantages, and editorial uniformity, a report published on a quarterly basis was recommended.

The *CDC Surveillance Summaries* contain information more reflective of the detailed surveillance reports of the past. CDC hopes that the *Surveillance Summaries* will disseminate surveillance data on a regular schedule, improve the clarity of community public health information, and also realize a cost savings. Although the *CDC Surveillance Summaries* are published quarterly, they will not be limited to quarterly data; annual data will probably be more typical. The *MMWR Annual Summary* will complement rather than serve as the cumulative summary of the quarterly publications.

Data Sources

Data on the reported occurrence of notifiable diseases are derived from reports supplied by the state and territorial departments of health and CDC program activities, routinely published in the *MMWR*, and compiled in final form in the *MMWR Annual Summary*.

CDC also maintains national surveillance programs for selected diseases with the cooperation of state and local health departments as well as other federal agencies, and publishes detailed epidemiologic analyses periodically. Data appearing in the *CDC Surveillance Summaries* or in a surveillance report may not agree exactly with reports published in the *MMWR* because of differences in timing of reports or because of refinements in case definition. It should be noted that data collected for the *MMWR* and the more detailed data published by individual CDC programs are collected independently.

These data should be interpreted with caution. Some diseases that cause severe clinical illness and are associated with serious consequences are probably reported quite accurately. However, diseases that are clinically mild and infrequently associated with serious consequences are less likely to be reported. Additionally, subclinical cases are seldom detected except in the course of epidemic investigations or special studies. The degree of completeness of reporting is also influenced by the diagnostic facilities available, the control measures in effect, and the interests and priorities of state and local officials responsible for disease control and surveillance. Finally, factors such as the introduction of new diagnostic tests and the discovery of new disease entities may cause changes in disease reporting independent of the true incidence of disease. Despite these limitations the data in these reports have proven to be useful in the analysis of trends.

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**Surveillance Programs
Centers for Disease Control**

Surveillance program	Responsible branch	Most recent report/summary*
Abortion	Pregnancy Epidemiology Branch Division of Reproductive Health Center for Health Promotion and Education	May 1983 (SS 32/2) (data from 1979-1980)
Behavioral risk factors	Division of Nutrition Center for Health Promotion and Education	SS 33/1 (data from 1981-1983)
Berylliosis cohorts: registry of disease and exposure	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	March 1983 (data from 1951-1980)
Biologics	Data Management Branch Division of Immunization Center for Prevention Services	Dec 1982 (1982 data)
Botulism	Enteric Diseases Branch Division of Bacterial Diseases Center for Infectious Diseases	May 1979 (data from 1899-1977)
Brucellosis	Bacterial Zoonoses Activity Division of Bacterial Diseases Center for Infectious Diseases	June 1979 (1978 data)
Coal workers' pneumoconiosis	Epidemiological Investigations Branch Division of Respiratory Disease Studies National Inst. for Occup. Safety & Hlth.	Feb 1983 (SS 32/1) (data from 1978-1980)
Congenital malformations	Birth Defects Branch Chronic Diseases Division Center for Environmental Health	Feb 1983 (SS 32/1) (data from 1970-1980)
Dengue	Dengue Branch Division of Vector-Borne Viral Diseases Center for Infectious Diseases	SS 33/1 (1982 data)
Diabetes	Division of Diabetes Control Center for Prevention Services	June 1979 (1978 data)
Diphtheria	Surveillance, Investigations and Research Branch Division of Immunization Center for Prevention Services	July 1978 (data from 1971-1975)

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible branch listed.

**Surveillance Programs
Centers for Disease Control**

Surveillance program	Responsible branch	Most recent report/summary*
Ectopic pregnancy	Pregnancy Epidemiology Branch Division of Reproductive Health Center for Health Promotion and Education	SS 33/2 (data from 1979-1980)
Encephalitis	Arbovirus Reference Branch Division of Vector-Borne Viral Diseases Center for Infectious Diseases	May 1981 (1978 data)
Enterovirus	Respiratory and Enterovirus Branch Division of Viral Diseases Center for Infectious Diseases	Nov 1981 (data from 1970-1979)
Fifteen leading causes of death in the U.S., 1978	Health Analysis and Planning for Preventive Services Center for Prevention Services	Sept 1982 (1978 data)
Food-borne disease	Enteric Diseases Branch Division of Bacterial Diseases Center for Infectious Diseases	June 1983 (1981 data)
Hepatitis	Hepatitis Laboratory Branch Division of Hepatitis and Viral Enteritis Center for Infectious Diseases	May 1983 (SS 32/2) (1981 data)
Homicide	Violence Epidemiology Branch Office of the Director Center for Health Promotion and Education	May 1983 (SS 32/2) (data from 1970-1978)
Hysterectomy	Epidemiologic Studies Branch Division of Reproductive Health Center for Health Promotion and Education	Aug 1983 (SS 32/3) (data from 1979-1980)
Influenza	Influenza Branch Division of Viral Diseases Center for Infectious Diseases	July 1984 (data from 1983-1984)
Lead poisoning in workers	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	April 1983 (data from 1976-1980)

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible branch listed.

**Surveillance Programs
Centers for Disease Control**

Surveillance program	Responsible branch	Most recent report/summary*
Leprosy	Respiratory and Special Pathogens Branch Division of Bacterial Diseases Center for Infectious Diseases	April 1976 (data from 1971-1973)
Leptospirosis	Bacterial Zoonoses Activity Division of Bacterial Diseases Center for Infectious Diseases	Aug 1979 (1978 data)
Malaria	Malaria Branch Division of Parasitic Diseases Center for Infectious Diseases	Aug 1983 (SS 32/3) (data from 1978-1982)
Maternal mortality	Division of Reproductive Health Center for Health Promotion and Education	SS 33/1 (data from 1974-1978)
Measles	Surveillance, Investigations and Research Branch Division of Immunization Center for Prevention Services	Sept 1982 (data from 1977-1981)
Mumps	Surveillance, Investigations and Research Branch Division of Immunization Center for Prevention Services	July 1978 (data from 1974-1976)
National electronic injury surveillance system	Safety Surveillance Branch Division of Safety Research National Inst. for Occup. Safety & Hlth.	May 1983 (SS 32/2) (1982 data)
National Occupational Hazard Survey (NOHS)	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	NIOSH Technical Report DHHS (NIOSH) Pub. No. 83-117
Nosocomial infections	National Nosocomial Infections Study Hospital Infections Program Center for Infectious Diseases	SS 33/2 (1983 data)
Nutrition	Division of Nutrition Center for Health Promotion and Education	Nov 1982 (1980 data)
Occupational characteristics of disabled workers	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	July 1980 (data from 1969-1972)

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible branch listed.

**Surveillance Programs
Centers for Disease Control**

Surveillance program	Responsible branch	Most recent report/summary*
Occupational injuries among loggers	Safety Surveillance Branch Division of Safety Research National Inst. for Occup. Safety & Hlth.	Aug 1983 (SS 32/3) (data from 1969-1974)
Occupational mortality in working for State	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	DHHS (NIOSH) Pub. No. 83-116 (data from 1950-1979)
Pediatric nutrition	Division of Nutrition Center for Health Promotion and Education	SS 32/4 (1982 data)
Pelvic inflammatory disease	Division of Sexually Transmitted Disease Center for Prevention Services	SS 32/4 (data from 1965-1982)
Plague	Plague Branch Division of Vector-Borne Viral Diseases Center for Infectious Diseases	SS 33/1 (1983 data)
Poliomyelitis	Surveillance, Investigations and Research Branch Division of Immunization Center for Prevention Services	Dec 1982 (data from 1980-1981)
Psittacosis	Bacterial Zoonoses Activity Division of Bacterial Diseases Center for Infectious Diseases	Feb 1983 (SS 32/1) (1979 data)
Rabies	Viral and Rickettsial Zoonoses Branch Division of Viral Diseases Center for Infectious Diseases	Feb 1983 (SS 32/1) (1981 data)
Reye syndrome	Epidemiology Office Division of Viral Diseases Center for Infectious Diseases	SS 32/4 (data from 1981-1982)
Rickettsial disease (RMSF, murine typhus, Q fever, endemic typhus)	Viral and Rickettsial Zoonoses Branch Division of Viral Diseases Center for Infectious Diseases	May 1981 (1979 data)
Rubella	Surveillance, Investigations and Research Branch Division of Immunization Center for Prevention Services	May 1980 (data from 1976-1978)

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible branch listed.

**Surveillance Programs
Centers for Disease Control**

Surveillance program	Responsible branch	Most recent report/summary*
<i>Salmonella</i>	Enteric Diseases Branch Division of Bacterial Diseases Center for Infectious Diseases	Dec 1982 (1980 data)
Sentinel health event (occupational) (SHE)	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	Sept 1983
Summer mortality	Special Studies Branch Chronic Diseases Division Center for Environmental Health	Feb 1983 (SS 32/1) (data from 1979-1981)
Surgical sterilization	Epidemiologic Studies Branch Division of Reproductive Health Center for Health Promotion and Education	Aug 1983 (SS 32/3) (data from 1979-1980)
Trichinosis	Helminthic Diseases Branch Division of Parasitic Diseases Center for Infectious Diseases	SS 33/2 (1982 data)
Tuberculosis	Division of Tuberculosis Control Center for Prevention Services	Sept 1982 (1981 data) TB Statistics: States & Cities Nov 1983 (1980 data) TB in the United States
U.S. immunization survey	Surveillance, Investigations and Research Branch Division of Immunization Center for Prevention Services	April 1983 (data from 1979-1982)
Venereal disease	Division of Sexually Transmitted Disease Center for Prevention Services	(1980 data) Sexually Transmitted Diseases Statistical Letter-No. 130 (data from 1978-1979) STD Fact Sheet-Edition 35
Water-related disease outbreaks	Enteric Diseases Branch Division of Bacterial Diseases Center for Infectious Diseases	Aug 1983 (1982 data)

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible branch listed.

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Ectopic Pregnancy in the United States, 1979-1980

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Introduction

Ectopic pregnancy has become an important public health problem in the United States. The incidence of ectopic pregnancy increased almost three-fold in women ages 15-44 years from 1970-1980. Previous CDC publications (1-3) have reported data on ectopic pregnancy from 1970-1978; this report updates that information to include data from 1979 and 1980.

Methods

The National Hospital Discharge Survey (NHDS), conducted by the National Center for Health Statistics (NCHS), provided an estimate of the number of ectopic pregnancies nationwide using a systematic sample of medical records from approximately 400 representative nonfederal hospitals. The U.S. Bureau of the Census and the NCHS provided denominator data (i.e., numbers of women ages 15-44 and numbers of live births) to compute estimated rates of ectopic pregnancy. Data on induced abortion were obtained from the CDC abortion surveillance system. The NCHS reported ectopic pregnancy deaths for the years 1970-1978. CDC began active epidemiologic surveillance of ectopic pregnancy deaths in 1979, relying primarily on information from State health departments.

The rates of ectopic pregnancy were obtained by dividing the estimated number of ectopic pregnancies by three annual estimates: 1) the female population ages 15-44, 2) live births, and 3) reported pregnancies (defined as the sum of live births, legal induced abortions and ectopic pregnancies). These annual rates for 1970-1980 are shown in Table 1. For brevity, the remaining tables in this summary list rates by reported pregnancies only. The four

TABLE 1. Number and rate of ectopic pregnancies, by year, United States, 1970-1980

Year	Number	Females ages 15-44*	Live births†	Reported pregnancies‡
1970	17,800	4.2	4.8	4.5
1971	19,300	4.4	5.4	4.8
1972	24,500	5.5	7.5	6.3
1973	25,600	5.6	8.2	6.8
1974	26,400	5.7	8.4	6.7
1975	30,500	6.5	9.8	7.6
1976	34,600	7.2	11.0	8.3
1977	40,700	8.3	12.3	9.2
1978	42,400	8.5	12.8	9.4
1979	49,900	9.9	14.3	10.4
1980	52,200	9.9	14.5	10.5
Total	363,700	7.0	9.9	7.8

*Rate/10,000 females.

†Rate/1,000 live births.

‡Rate/1,000 reported pregnancies (live births, legal induced abortions, and ectopic pregnancies).

geographic regions used on some of the tables are defined by the U.S. Bureau of the Census. Women were grouped and analyzed as "white" or "black", the black group including all races other than white. Estimates of ectopic pregnancy occurrence have been rounded to the nearest hundred. This rounding sometimes causes the sum of individual numbers not to equal the total. Rates were calculated from the unrounded estimates.

Results

For the period 1970-1980, a total of 363,700 ectopic pregnancies were reported with an overall rate of 7.8/1,000 reported pregnancies. The annual increase in the numbers and rates continued in 1979 and 1980 (Table 1). The number of reported ectopic pregnancies rose from 42,400 in 1978 to 49,900 in 1979 to 52,200 in 1980. This represents an annual increase of 11.0% between 1978 and 1980 compared with an annual increase of 11.5% between 1970 and 1978. The rates of ectopic pregnancy/1,000 reported pregnancies rose from 9.4 in 1978 to 10.4 in 1979 to 10.5 in 1980.

The risk of ectopic pregnancy increased with age (Table 2). Women in the 25-34 year age group had 53% of the ectopic pregnancies. While only 11% of the ectopic pregnancies occurred among women in the 35-44 year age group, this group had the highest rate of ectopic pregnancy/1,000 reported pregnancies.

Black women were at increased risk for ectopic pregnancy. While white women had 71% of the ectopic pregnancies, the rates were higher for black women in each age group and were almost double the rate for white women in the 25-34 and 35-44 year age groups (Figure 1).

The four geographic regions had similar overall rates of ectopic pregnancy, with the highest rates occurring in the Northeast and the lowest in the South. The rates increased with age in each of the geographic regions (Table 3). The rates of ectopic pregnancy for white women were lowest in the South and for black women in the West (Table 4). The highest rates for white women occurred in the Northeast and West, while the highest rates for black women were in the North Central region.

TABLE 2. Number and rate of ectopic pregnancies, by age and race,* United States, 1970-1980

Number of ectopic pregnancies			
Age	White	Black	Total
15-24	93,700	37,300	131,000
25-34	137,700	55,100	192,800
35-44	25,400	14,500	39,900
Total	256,800	106,900	363,700
Rate/1,000 reported pregnancies (live births, legal induced abortions, and ectopic pregnancies)			
Age	White	Black	Total
15-24	4.8	6.1	5.1
25-34	9.1	17.6	10.5
35-44	13.2	26.0	16.0
Total	7.0	10.9	7.8

*Unknown race redistributed according to the percentage of race known.

The mean length of hospital stay for ectopic pregnancies declined from 7.2 days in 1970 to 5.8 days in 1977, but remained essentially unchanged after 1977 (Table 5). Despite the shorter mean hospital stay in 1980, the estimated number of days women with ectopic pregnancy were hospitalized increased between 1978 and 1980 because of the increased incidence.

FIGURE 1. Ectopic pregnancy rates per 1,000 reported pregnancies (including live births, legal induced abortions, and ectopic pregnancies), by age and race, United States, 1970-1980

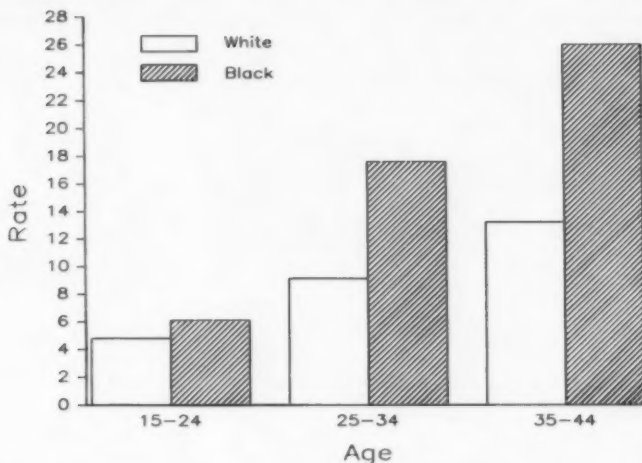


TABLE 3. Number and rate of ectopic pregnancies, by age and geographic region, United States, 1970-1980

Number of ectopic pregnancies				
Age	Northeast	North Central	South	West
15-24	27,700	34,300	42,700	26,300
25-34	48,400	51,800	55,700	36,900
35-44	11,700	6,900	12,500	8,900
Total	87,800	93,000	110,800	72,100

Rate/1,000 reported pregnancies (live births, legal induced abortions, and ectopic pregnancies)				
Age	Northeast	North Central	South	West
15-24	5.2	5.4	4.9	5.0
25-34	10.7	10.9	10.4	9.9
35-44	17.7	11.7	17.9	18.9
Total	8.4	7.9	7.5	7.6

Ectopic pregnancy has become an important cause of maternal mortality. Deaths from ectopic pregnancy represented 14% of all maternal deaths in 1980 (4). During the period 1970-1980, 524 women were reported to have died from ectopic pregnancies; 39 women died in 1979 and 46 women died in 1980. The overall death-to-case rate dropped from 0.9 deaths/1,000 ectopic pregnancies in 1978 to 0.8 in 1979, and rose again to 0.9 in 1980 (Table 6). The mortality rate is similar in all age groups with an overall rate from 1970-1980 of 1.4 deaths/1,000 ectopic pregnancies (Table 7). Mortality rates were highest in the South and lowest in the West.

Black women continue to have higher mortality rates than white women in all age categories (Figure 2). The overall death-to-case rate for black women was 3.6 times that of white women.

TABLE 4. Number and rate of ectopic pregnancies, by race* and geographic region, United States, 1970-1980

Number of ectopic pregnancies				
Race	Northeast	North Central	South	West
White	61,200	69,100	68,000	60,700
Black	26,600	23,900	42,800	11,400
Total	87,800	93,000	110,800	72,100
Rate/1,000 reported pregnancies (live births, induced abortions, and ectopic pregnancies)				
Race	Northeast	North Central	South	West
White	7.5	7.0	6.4	7.5
Black	11.8	13.1	10.3	8.8
Total	8.4	7.9	7.5	7.6

*Unknown race redistributed according to the percentage of race known.

TABLE 5. Number of ectopic pregnancies and average length of hospital stay for females ages 15-44, United States, 1970-1980

Year	Number	Average length of stay (days)	Total person-days hospitalized
1970	17,800	7.2	127,800
1971	19,300	7.1	136,700
1972	24,500	7.1	173,800
1973	25,600	7.4	189,200
1974	26,400	6.8	179,200
1975	30,500	6.1	186,200
1976	34,600	6.4	221,600
1977	40,700	5.8	236,200
1978	42,400	5.9	249,900
1979	49,900	5.9	294,300
1980	52,200	5.8	302,600
Total	363,700	6.4	2,297,500

Discussion

There was a 2.9-fold increase in the incidence of ectopic pregnancy between 1970 and 1980. Rates of ectopic pregnancy increased with age. Rates for black women were higher in every age category and geographic region than those for white women. Overall, black women had a 1.6 times greater risk of developing an ectopic pregnancy than did white women. Moreover, this risk rose with age, from a relative risk of 1.3 for black women ages 15-24 to a relative risk of 2.0 for black women ages 35-44. Since ectopic pregnancy rates rise with age, the combined effect of age and race was even greater. Black women ages 35-44 were 5.4 times more likely to experience an ectopic pregnancy than white women ages 15-24.

TABLE 6. Death-to-case rate* for women with ectopic pregnancies, by race[†] and year United States, 1970-1980

Year	White	Black	Total
1970	2.2	7.2	3.5
1971	1.5	7.5	3.2
1972	1.6	2.8	2.0
1973	1.5	2.2	1.8
1974	1.0	4.7	1.9
1975	0.9	3.5	1.6
1976	0.4	2.9	1.1
1977	0.5	2.4	1.1
1978	0.4	1.8	0.9
1979	0.5	1.6	0.8
1980	0.5	1.8	0.9
Total	0.8	2.9	1.4

*Deaths from ectopic pregnancy/1,000 ectopic pregnancies.

[†]Race "unknown" was redistributed according to the known percentages for race.

TABLE 7. Number and rate of ectopic pregnancy deaths, by age, race and geographic region, United States, 1970-1980

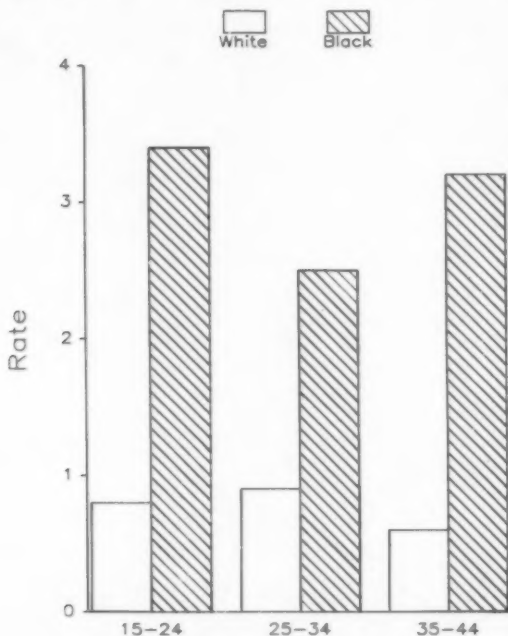
Characteristics	Number	Rate*
<i>Age:</i>		
15-24	200	1.5
25-34	262	1.4
35-44	60	1.5
<i>Race:</i>		
White	213	0.8
Black	309	2.9
<i>Geographic region:</i>		
Northeast	123	1.4
North Central	123	1.3
South	202	1.8
West	74	1.0
Total	522	1.4

*Deaths/1,000 ectopic pregnancies.

Concurrent with the rise in ectopic pregnancy rates, there was a rapid decline in the death-to-case rates (Figure 3). However, while the death-to-case rates were similar for all age groups and geographic regions, there was a marked difference between the rates for black and white women. Black women had a risk of death from ectopic pregnancy 3.6 times that of white women. Considering both the increased incidence of ectopic pregnancies among black women and their higher death-to-case rate, a pregnant black woman was 5.8 times as likely as a pregnant white woman to die from an ectopic pregnancy.

The total number of ectopic pregnancies may be underestimated because the NHDS data do not include figures from federal hospitals. In addition, an unknown number of ectopic pregnancies resolve spontaneously and remain undiagnosed. The number of reported pregnancies is also an underestimate, because it does not include spontaneous abortions or stillbirths, and the number of legal, induced abortions reported to CDC is consistently lower than the number reported by the Alan Guttmacher Institute. Given this underestimation of both numerator and denominator data, it is likely that the actual ectopic pregnancy rates are somewhat different from the reported rates. Because denominator data are probably underestimated to a greater extent than numerator data, the error is most likely toward overestimation of rates.

FIGURE 2. Death-to case rates* for women with ectopic pregnancies, by age and race, United States, 1970-1980



*Deaths from Ectopic Pregnancy per 1,000 Ectopic Pregnancies

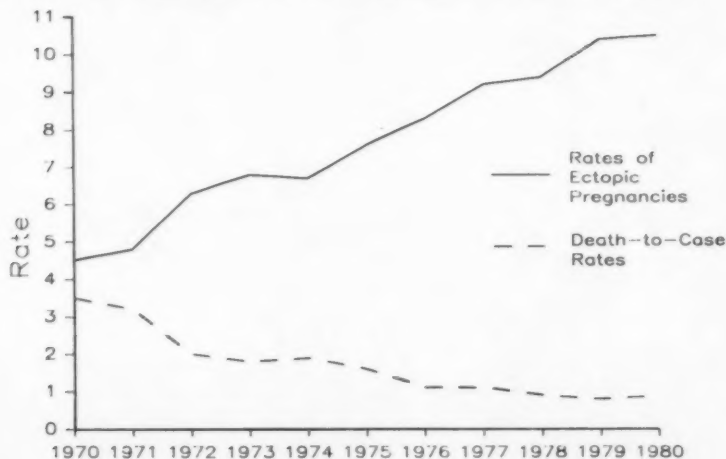
The leveling of the ectopic pregnancy death rate over the years 1978-1980 may reflect more complete ascertainment of such deaths. Deaths were reported directly to CDC by State health departments for the first time during 1979 and 1980; and this active surveillance system may have resulted in more complete reporting than was possible before.

The increase in ectopic pregnancy incidence during the period 1970-1980 may be related to the increase in pelvic inflammatory disease that has occurred during the past two decades in the United States (5). A higher incidence of sexually transmitted disease and higher rates of hospitalization for pelvic infection in black women may account for the higher ectopic pregnancy rates seen in black women (6, 7). Differences in the availability or the utilization of medical care may account for higher death-to-case rates among black women (8). The overall drop in the death-to-case rates is probably due to improved diagnosis and medical management.

References

1. Centers for Disease Control. Ectopic Pregnancy Surveillance. In: *CDC Surveillance Summaries* (published four times a year). February 1983;32(Suppl. 1):19SS-21SS.
2. Centers for Disease Control. Ectopic Pregnancy Surveillance, 1970-1978. Issued July 1982.
3. Rubin GL, Peterson HB, Dorfman SF, et al. Ectopic Pregnancy in the United States 1970 through 1978. *JAMA* 1983;249:1725-9.
4. National Center for Health Statistics. Advance report, Final Mortality Statistics, 1980. Monthly Vital Statistics Report, Vol. 32-No. 4, Suppl. DHHS Pub. No. (PHS) 83-1120. Public Health Service, Hyattsville, MD. August 1983.
5. Curran JW. Economic consequences of pelvic inflammatory disease in the United States. *Am J Obstet Gynecol* 1980;138:848-51.
6. St. John RK, Jones OG, Blount JH, Zaidi AA. Pelvic inflammatory disease in the United States: Epidemiology and trends among hospitalized women. *Sex Transm Dis* 1981;8:62-6.
7. Darrow WW. Venereal infections in three ethnic groups in Sacramento. *Am J Public Health* 1976;66:446-9.
8. Dorfman SF. Deaths from ectopic pregnancy, United States, 1979 to 1980. *Obstet Gynecol* 1983;62:334-8.

FIGURE 3. Rates of ectopic pregnancies (per 1,000 reported pregnancies) and death-to-case rates (per 1,000 ectopic pregnancies), by year, United States, 1970-1980



Nosocomial Infection Surveillance, 1983

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Introduction

Nosocomial infections are an important cause of morbidity and mortality in hospitalized patients; approximately 5%-6% of hospitalized patients develop nosocomial infections (1). These infections result in a prolongation of hospitalization and cost over a billion dollars a year (2). The National Nosocomial Infections Study (NNIS) has collected and analyzed data on the frequency of nosocomial infections in United States hospitals since 1970. This report provides descriptive data on nosocomial infections in a sample of U.S. hospitals in 1983.

Materials and Methods

The methods of this study and characteristics of participating hospitals have been described in detail (3). In brief, hospitals participating in NNIS conduct active hospital-wide surveillance using uniform definitions of nosocomial infections. During 1983, 54 hospitals regularly (≥ 8 months) reported data to CDC. For each nosocomial infection detected, the following information was reported: site of infection, date of onset, whether the infection was associated with a surgical procedure, the pathogen(s) isolated, occurrence of secondary bacteremia, antimicrobial susceptibility of bacterial pathogens, service of the patient, and, for those patients who died with a nosocomial infection, the relationship of the infection to death. In addition, the hospitals reported the number of patients discharged each month from the six primary services: medicine, surgery, obstetrics, gynecology, pediatrics, and newborn.

Data are recorded on standardized forms, which are sent to CDC each month. When the data are received at CDC, they are coded, entered into a computer, and edited before being analyzed.

Results

The NNIS Sample. The hospitals participating in NNIS are not a probability sample of U.S. hospitals; however, the 54 hospitals that reported data regularly in 1983 range in size from 80 to over 1,200 beds, are located throughout the United States, and include hospitals owned by state and local governments, as well as by profit and non-profit organizations. The geographic distribution of the 54 hospitals among the four regions of the country (Northeast, North Central, South, West) is roughly the same as for all 6,053 U.S. hospitals included in the American Hospital Association Annual Survey of Hospitals (4). Hospitals affiliated with medical schools, referred to as teaching hospitals, are still greatly over-represented among the NNIS hospitals; 56% (30/54) of the NNIS hospitals are teaching hospitals, whereas only 14%

of the hospitals across the country are affiliated with medical schools. Similarly, the 54 NNIS hospitals tend to be large, with a median size of 407 beds, compared with a median size of only 110 beds for the 6,053 U.S. hospitals.

Despite these limitations, previous analyses have shown that data collected in NNIS can be usefully interpreted by stratifying the 54 reporting hospitals into three categories: 1) non-teaching hospitals, 2) small teaching hospitals (500 or fewer beds), and 3) large teaching hospitals (more than 500 beds) (3).

The infection rates (number of hospital-acquired infections/thousand patients discharged) were highest in the large teaching hospitals and lowest in the non-teaching hospitals (Table 1), as were the infection rates on each of the six services (Table 2). In all three categories of hospital, the infection rate was highest on the surgery service (SURG), followed generally by medicine (MED), gynecology (GYN), and obstetrics (OB). The one exception was in the small teaching hospitals, where the infection rates on the medicine and gynecology services were similar. Lowest infection rates were reported on the newborn (NEW) and pediatrics (PED) services.

The urinary tract was the most frequent site of infection, followed by surgical wounds and lower respiratory tract in all three hospital categories (Table 3). On each service and for each site of infection, the infection rates were highest in the large teaching hospitals and lowest in the non-teaching hospitals.

In all three hospital categories, infections of the urinary tract, surgical wounds, and lower respiratory tract accounted for over 70% of the infections (Table 4). Primary bacteremia accounted for a higher percentage of infections in the large teaching hospitals than in the other hospitals.

Combined Rates by Service and Site. In general, the site-specific infection rate within each service was highest in the large teaching hospitals and lowest in the non-teaching hospitals (Table 5). The site-specific infection rates by service show that for each hospital category, urinary tract infections occurred predominantly on the surgery, medicine, and gynecology

TABLE 1. Infection rates (cases/1,000 discharges), by hospital category, 1983

Hospital category	Infections	Discharges	Rate
Non-teaching	6,845	281,122	24.4
Small teaching	7,875	255,601	30.8
Large teaching	13,528	328,559	41.2
Total	28,248	865,282	32.7

TABLE 2. Infection rates (cases/1,000 discharges), by hospital category and service, 1983

Hospital category	SURG	MED	GYN	OB	NEW	PED
Non-teaching	32.1	27.8	13.5	10.3	8.9	2.2
Small teaching	42.6	35.0	35.6	15.6	11.0	11.0
Large teaching	57.5	47.5	31.4	16.9	18.4	16.8
Total	44.3	37.1	27.4	14.7	13.4	11.1

services. Surgical wound infections occurred predominantly on the surgery, obstetrics, and gynecology services. Lower respiratory infections occurred predominantly on the medicine and surgery services. Primary bacteremia occurred primarily on the surgery, medicine, and newborn services. Cutaneous infections occurred primarily on the newborn service.

Pathogens. Of the 28,248 infections reported, 66% were caused by single pathogens, and 19% were caused by multiple pathogens (Figure 1). No pathogen was identified in 5% of the infections, and no culture was obtained in 10%. Of the 85% of infections in which pathogens were identified, 86% were caused by aerobic bacteria, 2% by anaerobic bacteria, and 7% by fungi (Figure 1). Together viruses, protozoa, and parasites accounted for 5% of the infections of known etiology. *Escherichia coli*, *Staphylococcus aureus*, enterococci, and *Pseudomonas aeruginosa* were the most frequently reported pathogens (Table 6). *E. coli* was the most frequently reported pathogen on the medicine, surgery, obstetrics, and gynecology services; *S. aureus* was the most frequently reported pathogen on the pediatrics and newborn services. *P. aeruginosa* was the second most frequently identified pathogen on both the medicine and surgery services and was less frequent on the other four services; enterococci were the second most frequently identified pathogens on the obstetrics and gynecology services and third most frequently identified on the medicine and surgery services; coagulase-negative staphylococci were the second most frequently identified pathogens on the pediatrics and newborn services and fourth most frequently identified on the obstetrics service.

TABLE 3. Infection rates (cases/1,000 discharges), by hospital category and site of infection, 1983

Hospital category	UTI	SWI	LRI	CUT	BACT	Other
Non-teaching	11.1	4.0	4.1	1.3	1.3	2.5
Small teaching	13.0	6.3	4.6	1.5	1.7	3.9
Large teaching	15.0	7.0	7.5	2.7	3.8	5.2
Total	13.1	5.8	5.5	1.9	2.4	3.9

UTI=urinary tract infection

SWI=surgical wound infection

LRI=lower respiratory infection

CUT=cutaneous infection

BACT=primary bacteremia

TABLE 4. Percent distribution of infections at each of the major sites, by hospital category, 1983

Site	Hospital category			
	Non-teaching	Small teaching	Large teaching	Total
UTI	45.8	42.2	36.3	40.2
SWI	16.1	20.4	16.8	17.7
LRI	16.5	14.8	18.4	16.9
BACT	5.8	5.3	9.1	7.3
CUT	5.7	4.8	6.6	5.9
Other	10.1	12.5	12.7	12.0

E. coli was the pathogen most frequently associated with urinary tract infections, followed by enterococci, *P. aeruginosa*, *Klebsiella* spp., and *Proteus* spp. (Table 7). *S. aureus* was the pathogen most frequently associated with surgical wound infections, followed by enterococci, *E. coli*, coagulase-negative staphylococci, and *P. aeruginosa*. *P. aeruginosa* was the pathogen most frequently associated with lower respiratory tract infections, followed by *S. aureus*, *Klebsiella* spp., *Enterobacter* spp., and *E. coli*. Coagulase-negative staphylococci were the pathogens most frequently associated with primary bacteremia, followed by *S. aureus*, *E. coli*, *Klebsiella* spp., and enterococci.

When the pathogens causing infections at the five major sites were examined by service, interesting differences were noted (Table 8). On the surgery and medicine services, *E. coli*, *P.*

TABLE 5. Site-specific infection rates (cases/1,000 discharges), by service, 1983

1. Non-teaching hospitals

Service	Site						All sites
	UTI	SWI	LRI	BACT	CUT	Other	
Surg	13.3	8.9	5.1	1.3	1.2	2.3	32.1
Med	15.3	0.4	5.6	2.2	1.3	3.1	27.8
Gyn	8.2	3.8	0.4	0.2	0.2	0.7	13.5
Ob	2.8	4.1	0.2	0.2	0.3	2.7	10.3
Ped	0.0	0.5	0.5	0.1	0.3	0.8	2.2
New	0.3	0.1	1.4	1.3	3.8	2.2	8.9
Total	11.1	4.0	4.1	1.3	1.4	2.5	24.4

2. Small teaching hospitals

Service	Site						All sites
	UTI	SWI	LRI	BACT	CUT	Other	
Surg	16.0	13.5	6.6	1.6	1.0	3.9	42.6
Med	19.1	0.7	6.7	2.6	1.5	4.5	35.0
Gyn	20.6	11.8	0.8	0.4	0.3	1.6	35.6
Ob	3.4	8.0	0.7	0.3	0.4	2.7	15.6
Ped	1.4	0.6	1.3	1.6	1.1	5.0	11.0
New	0.4	0.2	0.8	1.3	4.8	3.4	11.0
Total	13.0	6.3	4.6	1.7	1.5	3.9	30.8

3. Large teaching hospitals

Service	Site						All sites
	UTI	SWI	LRI	BACT	CUT	Other	
Surg	19.2	15.5	10.1	4.3	2.9	5.5	57.5
Med	21.1	1.3	10.2	5.5	2.9	6.4	47.5
Gyn	16.2	7.7	3.0	1.1	0.7	2.2	31.4
Ob	4.7	7.5	0.4	0.7	0.6	3.0	16.9
Ped	2.2	2.4	3.2	2.4	2.3	4.4	16.8
New	0.7	0.4	4.0	2.6	5.5	5.2	18.4
Total	15.0	7.0	7.5	3.8	2.7	5.2	41.2

aeruginosa, enterococci, and *S. aureus* were the pathogens most frequently isolated. On the gynecology and obstetrics services, *E. coli* and enterococci were the most frequently identified pathogens. These were followed by *S. aureus* and *Klebsiella* spp. on the gynecology service and by group B *Streptococcus* and *S. aureus* on the obstetrics service. On the pediatrics and newborn services, *S. aureus*, coagulase-negative staphylococci, and *E. coli* were the most frequently isolated pathogens, followed by *Candida* spp., *Klebsiella* spp., and *P. aeruginosa* on the pediatrics service and by group B *Streptococcus*, *Klebsiella* spp., and *P. aeruginosa* on the newborn services. On all six services, *E. coli* was the pathogen most frequently isolated from the urinary tract, with enterococci second on the medicine, obstetrics, and gynecology services and *P. aeruginosa*, *Klebsiella* spp., and *Candida* spp. second on the surgery, newborn, and pediatrics services, respectively. *S. aureus* was the pathogen most frequently associated with surgical wound infections on all services except gynecology, where *E. coli* was isolated most frequently. The pathogen most frequently associated with lower respiratory infections varied by service; *P. aeruginosa* on the surgery and medicine services, *S. aureus* on the obstetrics and newborn services, and *Klebsiella* spp. on the gynecology and pediatrics services. Coagulase-negative staphylococci were most frequently associated with primary bacteremia on the pediatrics, newborn, and surgery services. In contrast, *S. aureus*, group B *Streptococcus*, and *Bacteroides* spp. were the pathogens most frequently associated with bacteremia on the medicine, obstetrics, and gynecology services, respectively.

Secondary Bacteremia. Secondary bacteremia was defined as a bloodstream infection with an organism that was also isolated from an infection at another site. Secondary bacteremia was reported most frequently by large teaching hospitals and least frequently by non-teaching hospitals (Table 9). Secondary bacteremia occurred most frequently on the pediatrics service, followed by the newborn and medicine services, and least frequently on the

FIGURE 1. Distribution of infections, by etiology, 1983

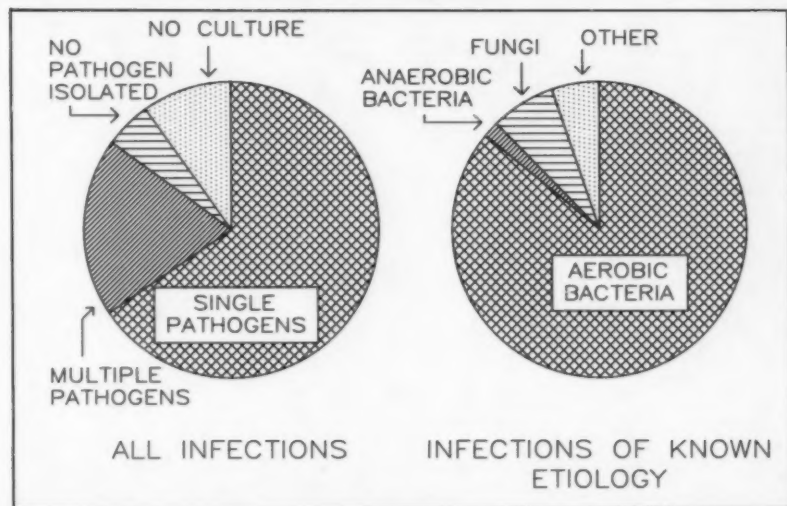


TABLE 6. The 15 most frequently isolated pathogens and their percentage distribution on each service, 1983

Pathogen	Med	Surg	Ob	Gyn	Ped	New	Total isolates	%
<i>E. coli</i>	21.3	16.3	20.6	32.5	11.4	7.7	5,779	18.6
<i>S. aureus</i>	8.9	10.8	9.5	6.9	16.8	34.1	3,356	10.8
Enterococci	9.9	10.9	15.3	17.2	5.4	6.3	3,308	10.7
<i>P. aeruginosa</i>	10.8	12.4	1.2	3.2	7.4	4.4	3,286	10.6
<i>Klebsiella</i> spp.	8.7	7.0	2.8	5.5	7.7	5.4	2,288	7.4
Coagulase-negative staphylococci								
<i>Enterobacter</i> spp.	5.1	6.1	6.7	4.9	12.4	14.6	1,892	6.1
<i>Proteus</i> spp.	5.2	7.3	2.0	3.7	1.5	2.1	1,811	5.8
<i>Candida</i> spp.	5.9	5.6	3.2	4.7	2.4	1.0	1,667	5.4
<i>Serratia</i> spp.	6.4	4.6	0.4	1.6	8.9	3.3	1,570	5.1
Other fungi	2.3	2.6	0.2	0.2	1.3	1.4	691	2.2
<i>Bacteroides</i> spp.	1.9	1.4	0.3	0.6	1.1	0.6	471	1.5
Group B	0.6	1.8	4.7	3.4	1.1	0.1	439	1.4
<i>Streptococcus</i>								
<i>Citrobacter</i> spp.	0.7	0.7	9.6	4.2	1.1	5.6	418	1.3
Other anaerobes	1.5	1.4	0.8	0.4	0.4	0.5	403	1.3
All others*	0.9	0.9	4.1	2.2	0.9	0.6	337	1.1
	9.9	10.2	18.6	8.8	20.2	12.3	3,296	10.6
Number of isolates	11,864	15,086	1,182	1,240	542	1,098	31,012	100.0

*No other pathogen accounted for more than 3% of the isolates on any service.

TABLE 7. The 15 most frequently isolated pathogens and their percentage distribution for each site of infection, 1983

Pathogen	UTI	SWI	LRI	BACT	CUT	Other	Total isolates	%
<i>E. coli</i>	31.7	11.4	7.1	9.5	7.7	7.3	5,779	18.6
<i>S. aureus</i>	1.6	19.0	12.8	12.8	33.3	16.4	3,356	10.8
Enterococci	14.9	11.4	1.6	7.3	9.5	7.3	3,308	10.7
<i>P. aeruginosa</i>	12.5	8.1	15.1	6.1	7.2	6.2	3,286	10.6
<i>Klebsiella</i> spp.	7.6	4.8	12.8	9.1	4.4	4.0	2,288	7.4
Coagulase-negative staphylococci								
<i>Enterobacter</i> spp.	3.7	8.4	1.1	14.2	9.5	11.1	1,892	6.1
<i>Proteus</i> spp.	4.4	6.9	10.0	6.9	4.1	4.1	1,811	5.8
<i>Candida</i> spp.	7.3	5.0	4.4	1.7	3.5	3.2	1,667	5.4
<i>Serratia</i> spp.	5.1	1.4	4.2	5.6	4.5	13.4	1,570	5.1
Other fungi	1.2	2.0	5.6	2.8	1.8	1.8	691	2.2
<i>Bacteroides</i> spp.	2.0	0.4	1.8	1.0	0.4	2.3	471	1.5
Group B	0.0	4.4	0.2	3.4	1.0	1.9	439	1.3
<i>Streptococcus</i>								
<i>Citrobacter</i> spp.	1.0	1.8	0.7	2.8	1.3	1.7	418	1.3
Other anaerobes	1.4	1.3	1.7	1.1	1.1	0.9	403	1.3
All others*	0.0	2.3	0.0	2.1	0.9	4.2	337	1.1
	5.6	11.4	20.9	13.6	9.8	14.2	3,296	10.6
Number of isolates	13,185	6,163	4,490	2,292	1,798	3,104	31,012	100.0

*No other pathogen accounted for more than 3% of the isolates at any site.



TABLE 8. Five most common pathogens isolated and percentage of t

Service	UTI		SWI		Pathogen
	Pathogen	%	Pathogen	%	
Medicine	<i>E. coli</i>	32.7	<i>S. aureus</i>	15.7	<i>P. aeruginosa</i>
	Enterococci	14.2	Enterococci	13.4	<i>S. aureus</i>
	<i>P. aeruginosa</i>	11.1	<i>E. coli</i>	11.1	<i>Klebsiella</i>
	<i>Klebsiella</i> spp.	8.2	<i>P. aeruginosa</i>	10.8	Enterobacter
	<i>Proteus</i> spp.	8.0	Enterobacter spp.	8.0	<i>E. coli</i>
Surgery	<i>E. coli</i>	28.6	<i>S. aureus</i>	19.6	<i>P. aeruginosa</i>
	<i>P. aeruginosa</i>	16.2	Enterococci	11.3	<i>Klebsiella</i>
	Enterococci	14.4	<i>E. coli</i>	10.9	Enterobacter
	<i>Klebsiella</i> spp.	7.3	<i>P. aeruginosa</i>	9.0	<i>S. aureus</i>
	<i>Proteus</i> spp.	7.1	Coag-neg Staph.	8.4	<i>E. coli</i>
Gynecology	<i>E. coli</i>	43.7	<i>E. coli</i>	17.2	<i>Klebsiella</i>
	Enterococci	20.2	<i>S. aureus</i>	16.6	<i>S. aureus</i>
	<i>Klebsiella</i> spp.	6.1	Enterococci	13.8	<i>P. aeruginosa</i>
	<i>Proteus</i> spp.	5.2	<i>Bacteroides</i> spp.	9.5	Enterobacter
	Group B Strep.	4.5	Coag-neg Staph.	7.4	<i>E. coli</i>
Obstetrics	<i>E. coli</i>	35.1	<i>S. aureus</i>	14.5	<i>S. aureus</i>
	Enterococci	27.0	<i>E. coli</i>	13.6	<i>E. coli</i>
	Group B Strep.	7.1	Enterococci	10.0	<i>Klebsiella</i>
	<i>Klebsiella</i> spp.	4.8	Group B Strep.	9.5	Enterococci
	<i>Proteus</i> spp.	4.5	Coag-neg Staph.	9.1	
Pediatrics	<i>E. coli</i>	38.6	<i>S. aureus</i>	28.8	<i>Klebsiella</i>
	<i>Candida</i> spp.	16.9	Coag-neg Staph.	16.4	<i>P. aeruginosa</i>
	<i>P. aeruginosa</i>	10.8	<i>E. coli</i>	8.2	<i>S. aureus</i>
	Enterococci	8.4	<i>P. aeruginosa</i>	6.8	<i>Candida</i>
	<i>Klebsiella</i> spp.	8.4	Enterococci	6.8	<i>E. coli</i>
Newborn	<i>E. coli</i>	25.5	<i>S. aureus</i>	30.4	<i>S. aureus</i>
	<i>Klebsiella</i> spp.	15.7	Coag-neg Staph.	26.1	<i>P. aeruginosa</i>
	Enterococci	9.8	Enterococci	13.0	Coag-neg
	<i>S. aureus</i>	7.8	<i>Klebsiella</i> spp.	13.0	<i>Klebsiella</i>
	Enterobacter spp.	5.9	<i>P. aeruginosa</i>	8.7	<i>E. coli</i>

Percentage of total within each site and service, 1983

Site					
LRI		CUT		BACT	
Pathogen	%	Pathogen	%	Pathogen	%
<i>Pseudomonas aeruginosa</i>	14.7	<i>S. aureus</i>	29.0	<i>S. aureus</i>	14.7
<i>S. aureus</i>	14.1	Coag-neg <i>Staph.</i>	9.7	Coag-neg <i>Staph.</i>	13.9
<i>Klebsiella</i> spp.	13.3	<i>P. aeruginosa</i>	8.9	<i>E. coli</i>	10.7
<i>Enterobacter</i> spp.	9.2	<i>Enterobacter</i> spp.	7.5	<i>Klebsiella</i> spp.	10.6
<i>E. coli</i>	7.5	<i>E. coli</i>	7.1	<i>P. aeruginosa</i>	7.5
<i>Pseudomonas aeruginosa</i>	15.6	<i>S. aureus</i>	19.2	Coag-neg <i>Staph.</i>	13.1
<i>Klebsiella</i> spp.	12.4	Enterococci	13.5	<i>S. aureus</i>	12.0
<i>Enterobacter</i> spp.	11.4	<i>P. aeruginosa</i>	10.0	<i>Enterobacter</i> spp.	10.3
<i>S. aureus</i>	11.1	Coag-neg <i>Staph.</i>	8.9	Enterococci	9.6
<i>E. coli</i>	6.9	<i>E. coli</i>	8.0	<i>E. coli</i>	8.8
<i>Klebsiella</i> spp.	17.2	Enterococci	25.0	<i>Bacteroides</i> spp.	14.7
<i>S. aureus</i>	17.2	<i>E. coli</i>	20.8	<i>Klebsiella</i> spp.	11.8
<i>Pseudomonas aeruginosa</i>	10.3	<i>S. aureus</i>	20.8	<i>E. coli</i>	8.8
<i>Enterobacter</i> spp.	10.3	<i>Proteus</i> spp.	8.3	Coag-neg <i>Staph.</i>	5.9
<i>E. coli</i>	3.4	<i>Enterobacter</i> spp.	4.2	<i>Proteus</i> spp.	5.9
Coag-neg <i>Staph.</i>	3.4				
Group B <i>Strep.</i>	3.4				
<i>S. aureus</i>	12.5	<i>S. aureus</i>	35.6	Group B <i>Strep.</i>	22.2
<i>E. coli</i>	8.3	<i>E. coli</i>	17.8	<i>S. aureus</i>	11.1
<i>Klebsiella</i> spp.	8.3	Coag-neg <i>Staph.</i>	11.1	Coag-neg <i>Staph.</i>	11.1
Enterococci	4.2	Enterococci	8.9	<i>Bacteroides</i> spp.	11.1
		<i>Proteus</i> spp.	6.7	Other anaerobes	8.9
		<i>Klebsiella</i> spp.	6.7		
<i>Klebsiella</i> spp.	16.7	<i>S. aureus</i>	35.2	Coag-neg <i>Staph.</i>	18.0
<i>Pseudomonas aeruginosa</i>	13.0	Coag-neg <i>Staph.</i>	14.8	<i>S. aureus</i>	12.4
<i>S. aureus</i>	13.0	Enterococci	8.0	<i>Klebsiella</i> spp.	10.1
<i>Candida</i> spp.	7.4	<i>E. coli</i>	6.8	<i>E. coli</i>	9.0
<i>E. coli</i>	3.7	<i>Candida</i> spp.	6.8	<i>Candida</i> spp.	6.7
		<i>P. aeruginosa</i>	6.8		
<i>S. aureus</i>	21.1	<i>S. aureus</i>	60.2	Coag-neg <i>Staph.</i>	22.0
<i>Pseudomonas aeruginosa</i>	16.7	Coag-neg <i>Staph.</i>	9.2	Group B <i>Strep.</i>	19.6
Coag-neg <i>Staph.</i>	12.3	<i>E. coli</i>	6.3	Enterococci	8.9
<i>Klebsiella</i> spp.	11.4	Enterococci	6.3	<i>S. aureus</i>	7.1
<i>E. coli</i>	7.0	<i>Klebsiella</i> spp.	2.9	<i>E. coli</i>	6.0

obstetrics and gynecology services. It was reported less frequently with infections of the urinary tract, surgical wounds, lower respiratory tract, and cutaneous infections than with infections, collectively, at "other" sites (Table 10). With respect to the five major sites, secondary bacteremia occurred least frequently following urinary tract infections. It occurred most frequently following infections caused by *Bacteroides* spp., *Serratia* spp., *S. aureus*, *Acinetobacter* spp., group B *Streptococcus*, and *Providencia* spp. (Table 11).

Antimicrobial Resistance. Resistance was defined as the number of resistant isolates divided by the number of organisms which were either sensitive or resistant. Methicillin-resistant *S. aureus* was most commonly reported from the large teaching hospitals (Table 12). Compared with 1980-1982, however, methicillin resistance in *S. aureus* increased in 1983 at non-teaching and small teaching hospitals and decreased slightly at large teaching hospitals (3). Similarly the proportion of *S. aureus* organisms resistant to gentamicin increased at small teaching hospitals but decreased at large teaching hospitals.

The percentages of *E. coli*, *K. pneumoniae*, *S. marcescens*, and *P. aeruginosa* organisms that were resistant to aminoglycosides and selected beta-lactam antibiotics varied according to the three hospital categories (Tables 13-16). Aminoglycoside resistance was most common in *P. aeruginosa* and *S. marcescens*, and cefotaxime or moxalactam resistance was most common in *P. aeruginosa*. Since 1982, amikacin resistance has increased for *K. pneumoniae* in all hospital categories (3).

TABLE 9. Percentage of infections* with secondary bacteremia, by service and hospital category, 1983

	Surg	Med	Gyn	Ob	New	Ped	All services
Non-teaching	3.8	5.1	1.2	3.7	1.1	0.0	4.2
Small teaching	4.4	6.3	0.6	2.6	3.0	6.7	4.7
Large teaching	6.5	7.5	2.6	3.6	9.9	12.1	6.8
All hospitals	5.2	6.5	1.6	3.2	6.0	9.6	5.5

*Excluding primary bacteremia.

TABLE 10. Percentage of infections with secondary bacteremia, by site* and hospital category, 1983

	Site					All sites
	UTI	SWI	LRI	CUT	Other†	
Non-teaching	3.2	4.6	4.0	4.6	7.6	4.2
Small teaching	2.9	4.4	5.6	4.2	10.8	4.7
Large teaching	3.8	6.0	5.9	6.6	17.5	6.8
All hospitals	3.4	5.2	5.4	5.6	13.5	5.5

*Excluding primary bacteremia.

†Most frequently associated with cardiovascular (55.3%), arterial (8.7%), intra-abdominal (8.1%), central nervous system (6.1%), and burn infections (4.6%).



TABLE 11. Ten pathogens with the highest percentage of associated

Pathogen	Non-teaching		Small teaching	
	Number of infections	Percentage with secondary bact.	Number of infections	Percentage with secondary bact.
<i>Bacteroides</i> spp.	24	16.7	65	13.1
<i>Serratia</i> spp.	123	9.8	115	10.4
<i>S. aureus</i>	648	7.7	828	8.1
<i>Acinetobacter</i>	26	0.0	27	0.0
Group B <i>Streptococcus</i>	57	7.0	87	8.1
<i>Providencia</i> spp.	14	7.1	13	7.7
Coagulase-negative				
<i>Staphylococcus</i>	212	4.2	356	6.1
<i>Enterobacter</i> spp.	285	5.3	321	4.4
<i>Klebsiella</i> spp.	366	4.4	404	4.4
<i>P. aeruginosa</i>	773	5.0	644	7.1

iated secondary bacteremia, by hospital category, 1983

teaching	Large teaching		All hospitals	
	Number of infections	Percentage with secondary bact.	Number of infections	Percentage with secondary bact.
Percentage with secondary bact.				
13.8	53	17.0	142	15.5
10.4	241	13.7	479	11.9
8.6	1,274	16.0	2,750	11.8
0.0	94	18.1	147	11.6
8.0	100	16.0	244	11.1
7.7	19	15.8	46	10.9
6.5	550	9.5	1,118	7.5
4.2	601	9.7	1,207	7.4
4.2	718	9.7	1,488	6.9
7.6	1,209	5.7	2,626	6.0

TABLE 12. Antimicrobial resistance of *S. aureus*, 1983

Hospital category	Number resistant (%)					
	Methicillin	Gentamicin	Clindamycin	Chloramphenicol	Erythromycin	
Non-Teaching	22 (4.1)	42 (8.1)	50 (7.8)	33 (6.0)	80 (12.3)	
Small teaching	32 (3.6)	37 (4.9)	49 (6.0)	24 (2.8)	88 (9.5)	
Large teaching	108 (7.8)	127 (12.3)	158 (10.6)	85 (6.4)	239 (15.9)	

TABLE 13. Antimicrobial resistance of *E. coli*, 1983

Hospital category	Number resistant (%)				
	Gentamicin	Tobramycin	Amikacin	Cefotaxime	Moxalactam
Non-Teaching	23 (1.8)	18 (1.6)	26 (2.5)	3 (3.0)	1 (2.2)
Small teaching	42 (2.3)	25 (2.1)	18 (4.0)	5 (5.0)	4 (7.1)
Large teaching	48 (2.1)	47 (2.0)	28 (2.2)	8 (1.8)	6 (1.8)

TABLE 14. Antimicrobial resistance of *K. pneumoniae*, 1983

Hospital category	Number resistant (%)				
	Gentamicin	Tobramycin	Amikacin	Cefotaxime	Moxalactam
Non-Teaching	13 (3.3)	7 (2.2)	12 (4.2)	2 (4.2)	2 (7.4)
Small teaching	16 (3.3)	9 (2.7)	6 (4.9)	3 (11.5)	1 (4.3)
Large teaching	111 (13.2)	93 (11.0)	16 (2.8)	14 (4.6)	3 (1.6)

TABLE 15. Antimicrobial resistance of *S. marcescens*, 1983

Hospital category	Number resistant (%)				
	Gentamicin	Tobramycin	Amikacin	Cefotaxime	Moxalactam
Non-Teaching	7 (5.3)	12 (10.8)	3 (3.0)	0 (0)	3 (10.7)
Small teaching	5 (4.1)	8 (7.9)	5 (8.9)	4 (10.5)	2 (7.4)
Large teaching	38 (12.7)	38 (13.3)	16 (10.4)	10 (12.5)	11 (10.9)

Mortality. Of the 54 NNIS hospitals, 50 (representing > 50% of hospitalized patients with fatal nosocomial infections) assessed and reported the relationship of infection to death. These 50 hospitals reported mortality data on a total of 26,096 infections. Approximately 1% of infections were reported to have caused death, and 3.6% were reported to have contributed to death (Table 17). Infections were more often reported to cause or contribute to death in the teaching hospitals.

Discussion

Nosocomial infections remain a significant cause of morbidity and mortality in hospitals in the United States. The National Nosocomial Infections Study is the only source of data collected prospectively on nosocomial infections from a group of U.S. hospitals. The nosocomial infections rate in NNIS hospitals during 1983 was 3.3 infections/hundred discharges, which is similar to the rate of infection reported for the 3-year period 1980-1982 (3). These data probably underestimate the true incidence of nosocomial infections in these hospitals; the Study on the Efficacy of Nosocomial Infection Control programs (SENIC Project) found that between 5% and 6% of hospitalized patients develop nosocomial infections (1). Many factors contribute to this underestimating, including variability in the intensity of surveillance and availability of laboratory support. Despite the use of standard definitions of nosocomial infections by infection control personnel, the intensity of the surveillance conducted in these hospitals varies. In addition, the intensity of surveillance varies by service and pathogen. Furthermore, detection of viral infections depends more on the availability of virology laboratory support than on surveillance; therefore, hospitals without such support will not detect most viral infections.

TABLE 16. Antimicrobial resistance of *P. aeruginosa*, 1983

Hospital category	Number resistant (%)									
	Gentamicin		Tobramycin		Amikacin		Cefotaxime		Moxalactam	
Non-Teaching	131	(17.5)	66	(9.3)	45	(7.7)	56	(72.7)	45	(38.8)
Small teaching	68	(9.3)	21	(3.5)	36	(10.8)	106	(51.2)	87	(46.5)
Large teaching	208	(16.2)	91	(6.7)	62	(6.3)	162	(44.5)	107	(26.5)

TABLE 17. Percentage of infections reported as having caused or contributed to death of the patient, 1983

Hospital category	Number of infections	Percentage that caused death	Percentage that contributed to death
Non-teaching	6,728	0.5	3.7
Small teaching	7,140	1.3	4.0
Large teaching	12,228	0.8	3.4
Total	26,096	0.9	3.6

Infection rates consistently increased from the non-teaching to the small teaching to the large teaching hospitals for all services and sites of infection, suggesting that this stratification of hospitals by hospital category effectively defines groups of patients who have different levels of risk for the development of nosocomial infections. This differential risk of infection is undoubtedly a by-product of severity of illness and the frequency of invasive diagnostic and therapeutic modalities. Data currently are not available specifically for intensive care units, but the rates of infection were highest on the surgery and medicine services, which have more high-risk patients, and lowest on the pediatrics and newborn services.

On the other hand, Valenti et al. have shown that viral nosocomial infections are more common in children than in adults (5). In addition, Welliver et al. have shown that viruses account for approximately 14% of nosocomial infections in the pediatric population in a hospital where viral cultures are done routinely (6). Since only a small proportion of NNIS hospitals have diagnostic virology laboratories, many viral infections probably go undetected. The failure to detect these infections may partially explain the lower nosocomial infection rates reported by NNIS hospitals on the pediatrics and newborn services. In addition, other factors, such as the short duration of stay by many pediatric patients and the frequent use of isolation precautions on the pediatric and newborn services, may reduce the incidence of nosocomial infections on these services.

Infection rates on different services and at different sites of infection within the three hospital categories varied little from those reported for 1982 (3). However, the increase in the primary bacteremia rates at non-teaching and large teaching hospitals identified during 1980-1982 continued through 1983, and a slight increase in the lower respiratory tract infection rate at non-teaching and large teaching hospitals emerged. Further study will be needed to identify the factors responsible for these increases.

Specimens for microbiologic testing were obtained from 90% of the patients reported to have nosocomial infections. For 85% of the nosocomial infections reported, an etiologic agent was identified; approximately 86% of these were aerobic bacteria. Fungi, parasites, and viruses were infrequently reported, reflecting in part the frequency with which these pathogens are looked for.

E. coli, *S. aureus*, enterococci, and *P. aeruginosa* were the four most common nosocomial pathogens. *E. coli* was the most frequently identified pathogen on the four adult services, reflecting the fact that this organism was the primary cause of urinary tract infections, which in turn were the most common type of infection on these services. *S. aureus* was the pathogen most frequently identified on the pediatrics and newborn services. Coagulase-negative staphylococci were the second most frequent cause of nosocomial infection on the pediatrics and newborn services and were an important cause of bacteremia on all services except gynecology. Further study will be required to assess the contribution of coagulase-negative staphylococci to the increasing rate of primary bacteremia, though recent studies suggest that the increasing use of long-line catheters may be responsible for this trend (7).

Previous analyses of NNIS data have suggested that secondary bacteremia carries an increased risk of death (3). Among the major sites of infection, cutaneous infections were most likely to result in secondary bacteremia, followed by lower respiratory, surgical wound, and urinary tract infections. Infections at sites other than these four major sites were, collectively, more frequently associated with secondary bacteremia. These include cardiovascular, arterial, intra-abdominal, and central nervous system infections. Because of the increased risk of death associated with secondary bacteremia, these infections should be a high priority for prevention and control.

Previous analyses have shown the incidence of methicillin-resistant *S. aureus* infections to

be greatest at large teaching hospitals (8). The 1983 NNIS data suggest that the incidence of methicillin-resistant *S. aureus* is now increasing in non-teaching and small teaching hospitals as well. Further study will be needed to discern the factors responsible for this increase.

Compared with 1980-1982, aminoglycoside resistance among *K. pneumoniae*, *S. marcescens*, and *P. aeruginosa* organisms also has increased in 1983 at non-teaching hospitals, and amikacin resistance has increased for all three organisms at the small teaching hospitals (3). The frequency of resistance to the third-generation cephalosporins, however, did not vary consistently by hospital category. The number of *K. pneumoniae* and *S. marcescens* specimens tested for resistance to cefotaxime and moxalactam was very small, so data regarding cefotaxime and moxalactam resistance in these organisms should be interpreted cautiously. Since 1982, both cefotaxime and moxalactam resistance among *P. aeruginosa* isolates has increased at non-teaching and small teaching hospitals. At the large teaching hospitals moxalactam resistance among *P. aeruginosa* isolates has increased, but cefotaxime resistance has decreased.

This nationwide nosocomial infection surveillance system is expanding in four directions. First, despite the usefulness of the 3-category stratification of hospitals used in this report, an infection-risk index identifying patients with different levels of risk of nosocomial infection is needed to permit more meaningful comparisons between hospitals. Infection rates at different institutions could then be compared within levels of patient risk or standardized for differences of distribution of patient risk. Second, microcomputer software to support an integrated nosocomial infection information management system in each hospital is being developed to improve the quality and timeliness of information collected in NNIS. Third, data on antimicrobial usage are needed to permit an assessment of the impact of antimicrobial usage on patterns of resistance in nosocomial pathogens. Finally, additional hospitals will be included in the surveillance system to provide data from a more representative sample of all acute-care hospitals in the United States.

References

1. Haley RW, Hooton TM, Culver DH, et al. Nosocomial infections in U.S. hospitals, 1975-1976. Estimated frequency by selected characteristics of patients. *Am J Med* 1981;70:947-59.
2. Haley RW. Preliminary cost-benefit analysis of hospital infection control programs (The SENIC Project). Proceedings of an International Workshop at Baiersbrunn, Germany, September 1977, pp.93-5.
3. Centers for Disease Control. Nosocomial Infection Surveillance, 1980-1982. In: *CDC Surveillance Summaries* (published four times a year). 1983;32(No. 4SS):1SS-16SS.
4. American Hospital Association: American hospital association guide to the health care field. Chicago: American Hospital Association, 1982.
5. Valenti WM, Hall CB, Douglas RG Jr, Menegus MA, Pincus PH. Nosocomial viral infections: 1. Epidemiology and significance. *Infection Control* 1980;1:33-7.
6. Welliver RC, McLaughlin S. Unique epidemiology of nosocomial infection in a children's hospital. *Am J Dis Child* 1984;138:131-5.
7. Haley CE, Gregory WW, Donowitz LG, et al. Neonatal intensive care unit (NICU) bloodstream infections (BSI): emergence of gram positive bacteria as major pathogens. Abstract 691. Presented at the 22nd Interscience Conference on Antimicrobial Agents and Chemotherapy, Miami Beach, FL, 1982 October 4-6.
8. Haley RW, Hightower AW, Khabbaz RF, et al. The emergence of methicillin-resistant *Staphylococcus aureus* infections in United States hospitals. *Ann Intern Med* 1982; 97:207-308.

Trichinosis Surveillance, 1982

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Introduction

Humans contract trichinosis by ingesting meat containing the encysted larvae of *Trichinella spiralis*. Clinical signs and symptoms are usually associated with exposure to a large inoculum of larvae and include fever, myalgia, periorbital edema, petechial hemorrhage, and eosinophilia. The disease can be fatal. Pork is the primary source of infection for humans; however, in recent years bears and other wild game have emerged as important sources of human disease. From 1947, when CDC surveillance activities for trichinosis began, through 1982, 7,627 cases of trichinosis were reported in the United States.

Materials and Methods

This report is based on information obtained from detailed trichinosis surveillance case report forms submitted on each case by state health departments to the Division of Parasitic Diseases, Center for Infectious Diseases, CDC. Supplemental data were obtained from records of the National Morbidity Reporting Service; reports of trichinosis serologic test results from the Helminthic Diseases Serology Laboratory, DPD; and investigations conducted by the DPD staff.

The accepted CDC case definition for trichinosis is as follows: 1) a *Trichinella*-positive muscle biopsy, 2) a positive titer on serologic examination, or a three-fold increase in titer noted between acute- and convalescent-phase serum specimens, or 3) compatible signs and symptoms, including periorbital edema, myalgia, petechial hemorrhage, and eosinophilia, when the patient has a history of ingestion of meat known to contain *Trichinella* larvae.

Results

In 1982, 95 cases of trichinosis in the United States were reported to CDC. There were 11 common-source outbreaks, which accounted for 36 (38%) of the total cases. There were no deaths reported, although the case-fatality ratio for the past 5 years (including 1982) has been 6.3 deaths/1,000 cases.

Geographic Distribution

Twenty-one states reported cases of trichinosis in 1982 (Table 1); however, 75% (71) of the cases were from eight states (New Jersey, Pennsylvania, Maryland, New York, Massachusetts, Illinois, Hawaii, and Colorado). The incidence of reported cases was 0.4/million population for the entire United States. The largest number of cases (23) was reported from New Jersey, but the states with the highest annual incidence were Vermont and Hawaii, with 5.8 and 5.0, cases/million population, respectively. (Cases are listed by the state where the patient ingested the implicated meat item if this information is known; otherwise, the case is listed by the patient's state of residence.)

For the 5-year period 1978-1982 (Figure 1), the highest mean annual trichinosis incidence (>1.9 cases/million population) was observed in Alaska (40.0 cases/million), Rhode Island (8.7), Connecticut (3.7), New Jersey (2.8), Louisiana (2.3), and Vermont (2.0). A moderately high mean incidence (1.1-1.9 cases/million population) was observed in Massachusetts, Hawaii, and Pennsylvania, which all had rates of 1.2/million. Incidence in each of the other states was <1.1 cases/million population; and in 15 states (Arkansas, Georgia, Iowa, Kentucky, Minnesota, Montana, Nebraska, Nevada, North Carolina, North Dakota, Oklahoma, South Dakota, Tennessee, Utah, Wyoming) and the District of Columbia, no cases were reported.

Distribution by Age and Sex

There were 42 cases in males and 53 in females in 1982. As in previous years the age distribution was similar for both sexes. The ages of patients ranged from 3 to 87 years, with a mean of 38 years (median 35). The mean age of male patients was 38 years (median 35) and that of female patients was 39 years (median 38).

Temporal Distribution

The only consistent seasonal pattern for trichinosis in the United States has been a peak in December and January, often related to common-source outbreaks associated with home-made pork sausage prepared for the Christmas holidays. In 1982 the incidence peaked in January, coincident with two common-source outbreaks in New Jersey involving 11 cases.

TABLE 1. Trichinosis cases by state, United States, 1982

State	Cases	Rate/million population*
New Jersey	23	3.0
Pennsylvania	9	0.8
Maryland	9	2.0
New York	8	0.5
Massachusetts	7	1.2
Illinois	6	0.5
Hawaii	5	5.0
Colorado	4	1.3
Missouri	3	0.6
Vermont	3	5.8
California	2	0.1
Connecticut	2	0.6
West Virginia	2	1.0
Texas	2	0.1
Oregon	2	0.8
New Hampshire	2	2.1
Ohio	2	0.2
Delaware	1	1.7
Alaska	1	2.3
Idaho	1	1.0
Indiana	1	0.2
Total	95	0.4

*Estimates of state populations on July 1, 1982.

Source: Bureau of Census, Current Population Series.

Source of Infection

The types of meat products implicated as the source of trichinosis in 1982 were known for 82 of the 95 cases. Pork products were implicated in 71 (86%). Of 66 cases for which the type of domestic pork product was specified, 46 (70%) involved sausage.

Nonpork products were implicated in 11 cases (13%). Ground beef was identified as the probable source of infection for four cases. Since cattle are strictly herbivorous and therefore not considered a natural reservoir of *T. spiralis*, it is probable that the ground beef was adulterated with pork. Infected bear meat was the source for seven cases in six states (Alaska, Idaho, New Hampshire, New York, Oregon, and Vermont).

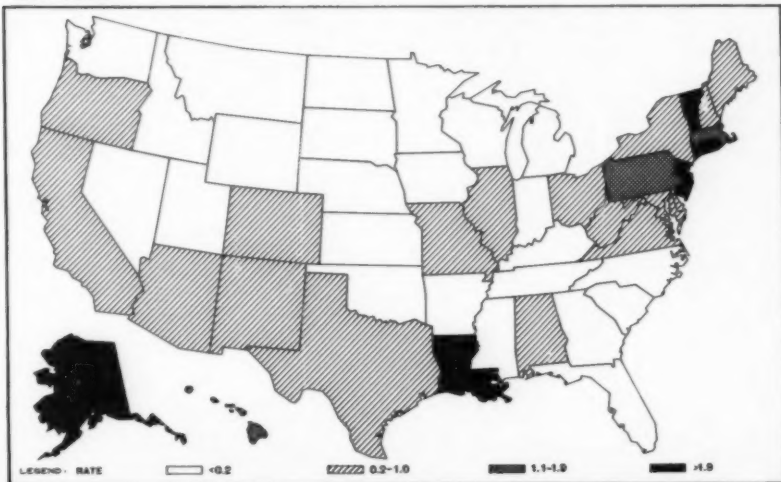
The place where the incriminated meat was obtained was reported for 76 cases. For 47 (60%) of these cases, the source was a supermarket, butcher shop, or other commercial outlet. Ten (13%) patients had consumed the incriminated meat items at a restaurant or other public eating place. Feral swine and bears obtained by hunting accounted for 12 (16%) cases. Seven (9%) cases were caused by pork from swine obtained directly from farms.

The method of cooking the incriminated meat was reported for 81 cases. For 57 (70%) of these, the meat had not been cooked. Reports on the other 24 cases indicated that the meat had been cooked (although apparently inadequately). Samples of the meat items believed responsible for 26 cases were examined by investigators for the presence of *T. spiralis*, and larvae were identified in 22 (85%).

Clinical Findings

Clinical information was reported for 85 cases; eosinophilia was detected in 95%, periorbital edema in 88%, fever in 94%, and myalgia in 97%. Information regarding gastrointestinal complaints and petechial hemorrhage was not requested. In the 57 cases for which the date of meat consumption is known, the mean incubation period was 15 days (range: 1-69 days).

FIGURE 1. Trichinosis mean annual incidence, by state and level of incidence, 1978-1982



From case reports for which biopsy and serologic test results were available, muscle biopsies were performed on 43 patients, and the results were positive for 37 (86%). Of 62 patients for whom serologic test results were reported, 51 (82.2%) were positive. The bentonite flocculation test was the most frequently used serodiagnostic test.

Discussion

A recent review of trichinosis surveillance data for 1947-1981 (1) discussed in detail the decline in the number of cases of trichinosis in the United States from >400 cases/year in 1947 to <150 cases/year observed over the last decade. Multiple factors account for this decline, most of them unrelated to planned trichinosis control measures. They include state and federal laws against feeding hogs raw garbage, which often contains the *Trichinella*-infected remains of livestock, game animals, or rodents.

These laws, which were designed to prevent the spread of highly contagious and economically devastating swine viral diseases, have also had an unplanned role in reducing trichinosis in swine. In addition, the widespread commercial and home freezing of pork, which kills trichinae, and increased consumer awareness concerning the need to cook pork products adequately have contributed to the reduction of this parasitic disease in the United States.

While proper curing of sausage destroys *Trichinella* larvae, making further preparation of the meat unnecessary, small processors and householders who prepare their own sausage are not always aware of established standards for the proper curing and cooking of pork products. Furthermore, the stamp "U.S. Inspected and Passed" on fresh raw pork products does not guarantee that the product is free from infective *Trichinella* larvae. U.S. Department of Agriculture specifications require that "ready-to-eat" pork products have been processed in a manner capable of destroying *Trichinella spiralis*. Methods specified in the regulations include heating, freezing, and curing procedures (2). The National Pork Producers Council recommends that pork roasts be cooked to an internal temperature of 170°F (77°C).

References

1. Schantz PM. Trichinosis in the United States—1947-1981. *Food Tech* 1983;37:83-6.
2. United States Department of Agriculture, Division of Food Safety and Quality Service (1973). Meat and poultry inspection regulations. Washington, D.C. p. 125.

Selected Bibliography

- CDC. Trichinosis Surveillance, 1981. In: *CDC Surveillance Summaries* (published four times a year). May 1983;32(Suppl. 2):15SS-22SS.
- CDC. Trichinosis Surveillance report—annual summary, 1980. Atlanta: CDC, October 1981.

Uses of Computer-Generated Maps in Occupational Hazard and Mortality Surveillance

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Introduction

In addition to identifying workplace hazards, the National Institute for Occupational Safety and Health (NIOSH) is responsible for developing a surveillance system that will detect and describe epidemiologically significant changes in work-related injury, disease, disability, and mortality. The scope of this surveillance system is broad and involves assessment of hazards and illnesses among 106 million American workers at nearly five million worksites over a land area of 3.5 million square miles (1). This paper describes the use of computer-generated maps as a surveillance technique for monitoring work-related hazards and mortality.

In the early 1960s, interest in the use of computer-generated maps for descriptive epidemiology began in the United States at the National Cancer Institute (NCI) (2). Researchers at NCI moved rapidly forward during the 1970s with the publication of maps depicting patterns of cancer mortality (3). At NIOSH, mapping work began as a result of support and collaboration from NCI. NIOSH shares with NCI, the National Center for Health Statistics (NCHS), and other research institutes, an interest in developing methods that allow the study of county-level profiles of mortality and occupational hazards.

Methods

NCHS compiles vital statistics from all States. County-level, cause-specific, mortality rates derived from these statistics have been used by investigators to map rates of cancer, cardiovascular disease, and infant mortality (4,5). NIOSH uses mortality data to map causes of death that are known or suspected to be associated with occupation. Other maps generate "leads" by identifying counties in which the workforce has a high potential for exposure to specific occupational hazards.

From 1972 to 1974, NIOSH conducted the National Occupational Hazard Survey (NOHS) and developed from this an inventory of chemical, physical, and biological hazards (6). A national probability sample of workplaces was selected in this survey for gathering information on the potential exposure of workers to hazards; 20 surveyors visited more than 4,500 industrial sites throughout the United States. Each site visit consisted of 1) a management interview to obtain basic information about the facility and its occupational health policies, and 2) a detailed walk-through survey of the plant. During the walk-through survey, all observed exposures to specific chemicals, tradename products, and physical agents were recorded as "potential exposures." The surveyors also noted the duration of potential exposures and any measures for industrial hygiene control that were being applied. The survey resulted in a data base of almost five million records describing potential workplace exposures by industry, occupation, and agent. This data base provided the first step for generating county-level maps of occupational hazards.

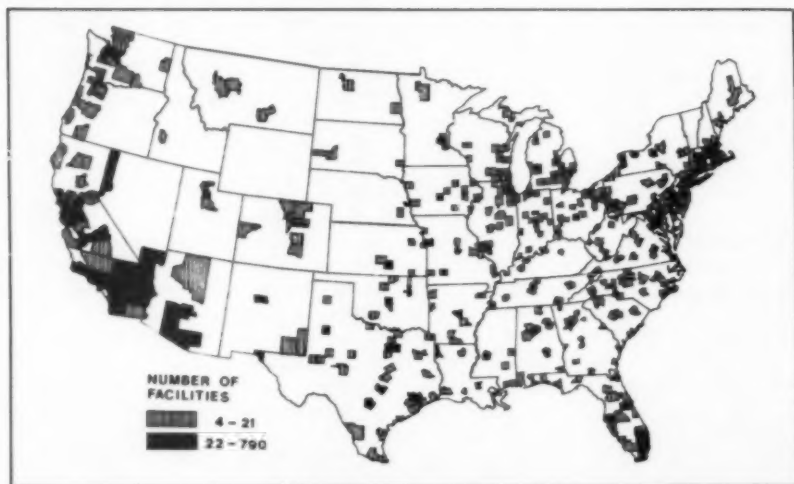
In the second step, a commercially available computerized file—the Dun & Bradstreet (D & B) file—was used to describe the characteristics of about 4.7 million American industrial facilities. The D & B descriptors include the Standard Industrial Classification (SIC) codes (7), the county in which the workplace is located, and the size of the workforce. The NOHS and D & B files have been linked according to SIC code. The types of potential exposures seen in the NOHS survey for a specified SIC code are assumed to be representative of all industries in the D & B file with the same SIC code. Using this linked data base, county-level maps are generated showing the approximate locations of worksites where there is a high probability of potential exposure to selected chemical agents.

NIOSH has developed five types of maps. The first four types depict potential exposures to a selected chemical agent that is thought to be a workplace hazard. The fifth type displays county-level, cause-specific mortality rates for a disease that may be related to this hazard.

Type 1. Worksites/specific hazard. Type 1 maps show the current locations of the types of worksites in which the specified agent was observed during the 1972-1974 NOHS. For example, Figure 1 shows a computer-generated map of the United States, coded to display, by county, the number of facilities (worksites) of the types where potential exposure to formaldehyde was observed in NOHS.

Two features of this map need explanation. First, the worksites represented on the map are from only those industries in which 10% or more of the workforce was found by the NOHS to be potentially exposed to formaldehyde (this is the "selection criterion"). Second, the shading of the counties on the map is determined by the number of these worksites located in each county. These numbers, in turn, are based on percentile distribution (≤ 60 , 61-89, and ≥ 90). Those counties containing three or fewer of the worksites in question are below the 60th percentile and are not shown on the map; those containing between four and 21 of

FIGURE 1. Distribution of facilities where workers are potentially exposed to formaldehyde (in 10% or more of the workforce)



the worksites fall between the 61st and 89th percentiles and are lightly shaded; and those containing 22 or more of the worksites are above the 90th percentile and have dark shading. Varying either the selection criterion or the criteria used to determine the shading will change the appearance of the map.

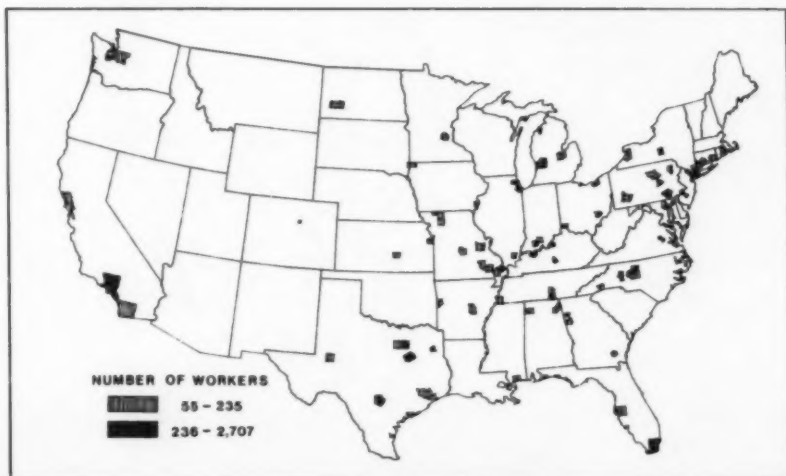
Type 2. Workers/specific hazards. Type 2 maps show the spatial distribution of workers, rather than worksites (Figure 2). Maps of this type may be used for identifying clusters of workers at risk of exposure to a specific industrial hazard. (Note that in Figure 2 the selection criterion is industries in which potential exposure was observed in 45% or more of the workforce—compared with 10% in Figure 1, and 5% in Figures 3 and 4.)

Type 3. Proportion of workers/hazard. Based on the Dun & Bradstreet count of workers in a given county, type 3 county-level maps are generated to estimate the proportion of the total workforce potentially exposed to a workplace hazard in a particular area. This estimate is useful in correlating occupational risks with county-based mortality patterns. Figure 3 shows the number of workers potentially exposed to formaldehyde/100,000 workers in the same area. These maps may suggest leads that warrant further epidemiologic investigation.

Type 4. Proportion of workers/hazard (state level). Another series of maps (type 4) is generated for individual states. These maps are useful in decentralizing occupational disease surveillance to the state level and helping State health departments improve their occupational health programs. For illustrative purposes, the State of Indiana has been selected to show the proportion of workers potentially exposed to formaldehyde (Figure 4).

Type 5. Cause-specific, county mortality rates. Surveillance efforts are concerned with workplace hazards and with work-related diseases, disabilities, and deaths. Geographic patterns for causes of death that may be related to occupational risks can be displayed in type 5 maps using standardized county-level, cause-specific mortality rates. In Figure 5, for example,

FIGURE 2. Distribution of workers potentially exposed to formaldehyde (in 45% or more of the workforce)



mortality rates for nasal cancer (thought to be related to formaldehyde exposure) are shown by county (8).

Discussion

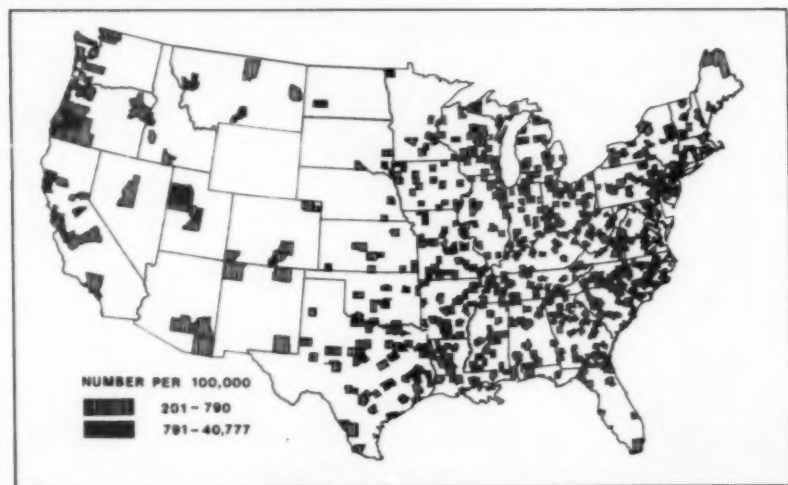
These maps represent efforts to present data on occupational hazards and mortality in a format for three general uses: 1) health-hazard and mortality maps can be used independently to generate occupational epidemiologic leads; 2) the juxtaposition of potential-hazard maps with maps showing causes of mortality may suggest hypotheses that warrant further study; and 3) computer-generated maps that use existing data files provide an efficient and effective means of disseminating information.

Much remains to be done methodologically. NIOSH is currently working to 1) increase the accuracy and completeness of the data on both hazards and mortality; 2) use more of the information that is shown on death certificates, (e.g., multiple cause of death coding and information on the industry and occupation of the deceased); 3) develop comparably coded county-level health parameters other than cause of death, (e.g., disease and disability data); and 4) produce and disseminate computer-generated maps in a way that will encourage their use in setting priorities. In addition, NIOSH is training and maintaining a staff skilled in the use of automated cartographic techniques.

References

1. U.S. Bureau of the Census. Statistical Abstract of the United States: 1981 (102nd edition). Washington, D.C., December 1981.
2. McKay FW. Automated cartography for cancer research. In: Proceedings of the 1976 workshop on automated cartography and epidemiology. Hyattsville, MD: Department of Health, Education and Welfare, 1979. (DHEW publication no [PHS] 79-1254).

FIGURE 3. Distribution of workers potentially exposed to formaldehyde (in 5% or more of the workforce)/100,000 employed



3. Mason TJ, McKay FW, Hoover R, Blot WJ, Fraumeni JF. Atlas of cancer mortality for U.S. counties 1950-1969. Washington, DC: Department of Health Education and Welfare, 1975. (DHEW publication no [NIH] 75-780).
4. Feinleib M, Fabsitz R. National Heart, Lung, Blood Institute mapping project. In: Proceedings of the 1976 workshop on automated cartography and epidemiology. Hyattsville, MD: Department of Health, Education, and Welfare, 1979. (DHEW publication no [PHS] 79-1254).
5. Kleinman, JC, Feldman JJ, Mugge RH. Geographic variation in infant mortality. Public Health Rep 1976; 91(5):423-32.
6. National Institute for Occupational Safety and Health. National occupational hazard survey. Volumes I-III survey manual. Cincinnati, OH: National Institute for Occupational Safety and Health (NIOSH), 1974, 1977, 1978. (DHEW publication nos. [NIOSH] 74-127, 77-213, 78-114).
7. Office of Management and Budget. Standard Industrial Classification Manual. Washington, D.C.: U.S. Government Printing Office, 1972.
8. National Institute for Occupational Safety and Health. Formaldehyde: evidence of carcinogenicity (Dec 23, 1980). Cincinnati, OH: National Institute for Occupational Safety and Health (NIOSH), 1981. (DHHS publication no [NIOSH] 81-111).

FIGURE 4. Distribution of workers potentially exposed to formaldehyde (in 5% or more of the workforce)/100,000 employed, in Indiana

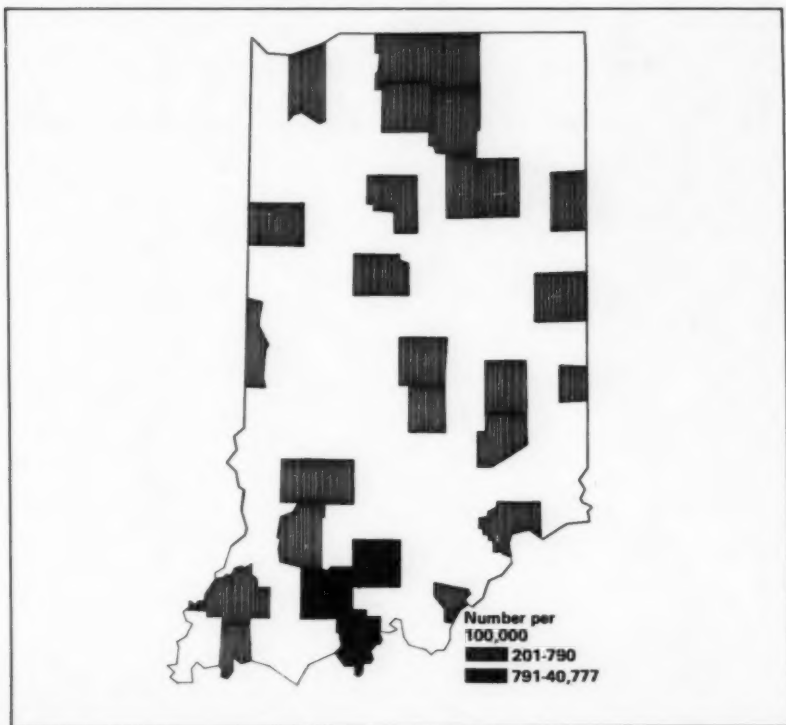
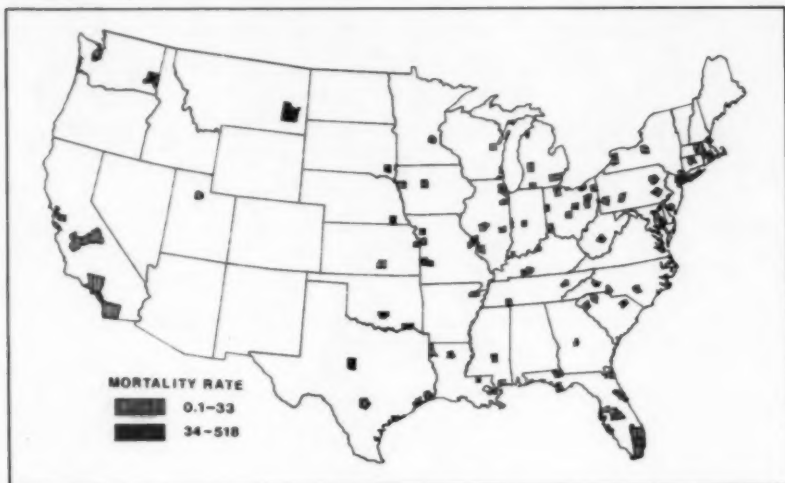


FIGURE 5. Deaths due to nasal cancer (ICDA 160), white males, ages 65 and over, 1972-1974



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The contributions of the State and Territorial Epidemiologists and the State Laboratory Directors to this report are gratefully acknowledged. The persons listed were in the positions shown as of August 1, 1984.

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